If Suicide Is a Public Health Problem, What Are We Doing to Prevent It?

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As for suicide . . . it is one of the leading causes of death in the world.

World Health Organization, 2002[1]

Although not a disease, suicide is a tragic endpoint of complex etiology and a leading cause of death worldwide.

Just as preventing heart disease once meant that specialists treated myocardial infarctions in emergency care settings, in the past decade, suicide prevention has been viewed as the responsibility of mental health professionals within clinical settings. By contrast, over the past 50 years, population-based risk reduction approaches have been used with varying levels of effectiveness to prevent morbidity and mortality associated with heart disease.

We examined whether the current urgency to develop effective interventions for suicide prevention can benefit from an understanding of the evolution of population-based strategies to prevent heart disease. (*Am J Public Health.* 2004;94:37–45)

Worldwide, there has been a call to reduce the substantial mortality and morbidity burden associated with suicide and suicidal behavior through sweeping, national strategies.[2–5] This development comes within an environment where there have been meager public health attempts to reduce these burdens, even while the limitations of high-risk approaches have been noted for some time.[6] Suicide prevention has narrowly focused on identifying proximate, individual-level risk factors, rather than thinking about population mental health in terms of complex social and ecological relations.[7] In 1969, at a time when the epidemiology of the risk factors for cardiovascular disease (CVD) was just beginning to be discovered (and debated), Caroline Bedell Thomas observed that "in both suicide investigations and cardiovascular studies, lifetime habits and personality factors are brought into focus as predictors of disease and death," and that "certain precursors of suicide, accident, fatal heart attack and fatal stroke *are already present and can be identified in youth*, many years before the event."[8(p282–283)]

Thomas further noted that while suicide prevention was in its infancy, the "preventive approach" already had been found to be effective at reducing the incidence of the most frequent cause of premature death, coronary heart disease. Public health approaches to reduce incidence coupled with clinically oriented efforts to prevent death from CVD have made significant advances in some populations since 1969, evidenced in the United States by the decline in the incidence of heart disease between 1970 and 1990 and significant reductions in mortality.[9] Prevention of CVD stands as an example of how clinicians and epidemiologists collaboratively approached overcoming the limitations of applying a purely biomedical approach to a disease whose origins are largely societal. This collabortion reflects a basic principle of the population risk reduction approach, one that many feel remains viable for preventing CVD despite the observation that declines in incidence rates stagnated during the 1990s.[10] If the basis of CVD is social and economic, the solution to the CVD epidemic has to be social and economic.[11,12]

Comparatively speaking, there have not been similar reductions in rates of depression or violence that potentially would contribute to preventing deaths due to suicide. It has only been in the past decade in the United States, with resolutions in Congress and reports from the surgeon general, that suicide prevention has been widely recognized as a problem requiring national attention and urgent action. While not a disease with a well-defined disease mechanism, suicide is nonetheless an extraordinarily adverse outcome. It reflects diverse risk factors and, like heart disease, is best understood within a complex paradigm of social, behavioral, and psychiatric factors. To the extent that efforts to reduce CVD and its precursors in some populations have risen to the challenge of preventing a disease that, like suicide, is the result of complex population processes, interdependencies, and multilevel causality, we consider these efforts to be a useful model for suicide prevention. We revisit the question raised by Thomas of whether we can learn to prevent suicide, but now within the (albeit imperfect) framework of prevention of CVD during the last 50 years.

WORLDWIDE VARIABILITY IN RATES OF SUICIDE

Analogous to diverse patterns of CVD, stroke, and hypertension worldwide, there is substantial worldwide variation in population patterns of suicide, violence, and depression.[13] As has been found for CVD, personality predispositions and psychological states may increase an individual's risk of complete suicide, which then has an impact on a population's pattern of suicide, interpersonal violence, and depression. A comprehensive understanding of this variability, accomplished through a deeper knowledge of the contributions of psychological and biological factors during different stages in the life course of individuals in different populations, could be critical for developing prevention programs.

For example, the unique pattern of suicide in China is recognized worldwide and is quite distinctive from Western trends in that more women kill themselves than men, commonly through poisoning from pesticides.[14] Studies testing models of how culturally specific social and environmental factors influence population health could potentially determine if this pattern results from the lower status of

women in China, beginning early in childhood, which is exacerbated by access to highly lethal means in adult life. Another illustrative example is that the higher than average suicide rates in many native and aboriginal communities in New Zealand, Canada, and the United States appear to be associated with early substance use,[15] but how this behavior may reverberate into adult life to cause higher rates of depression in these populations is unknown. A pattern of lower suicide rates among African Americans in the United States has been observed,[16] but it is potentially biased by differential case recognition. We suggest this because there are no studies that can explain this allegedly protective effect of being African American, particularly within the context of other violent behaviors (e.g., homicide) in some African American communities.[17]

The recent World Health Organization report notes that persistent social unrest in the states of the former Soviet Union is now taking its toll in terms of increased rates of depression and suicide, especially among men.[1] Greater industrialization among previously less-developed nations presents a disturbing picture of an upward slope in risk for suicide that may relate to a decrease in social ties and increased pressure to achieve in the workplace.[18] In Japan, high rates of suicide among men in their middle years may result from the interaction of long-held cultural beliefs concerning suicide and loss of social status following unemployment.[19] In the United States, evidence is mounting that an increase in the number of suicides in some university communities may represent only the tip of the iceberg of an epidemic of self-injurious behaviors and suicidal ideation.[20]

Be they temporal or cross-national, these very limited examples of the wide variation worldwide in patterns of suicide rates probably relate to fundamental differences in culturally based values and practices, or to major social forces such as war and macroeconomic issues. Social determinants of suicide are likely to contribute as much as, if not more than, individual risk factors, but they have been poorly studied to date. Indeed, an understanding of the collective characteristics of communities that may confer risk, at the individual level, for suicide and a whole family of outcomes has not advanced significantly since Durkheim's work in the 19th century,[21] although Hawton and colleagues' study on social class and suicidal behavior[22] comes the closest to doing so. But as McMichael[7] has pointed out, epidemiologists frequently misuse the sociological term "ecologic" to describe a study in which measurements are averaged over individuals. He suggests that a more accurate use of the term is one that depicts a model of the interdependencies between individuals, groups of humans, and their environments.

As an example, Leavey[23] misused the term "ecologic model" to describe the role of social cohesion and integration among Irish immigrants and risk of suicide. He concluded that population risk translated to enhanced risk of suicide at the individual level. But others[24] have pointed out that Leavey did not take into account that Irish migration into Britain is extremely heterogeneous, or attempt to account for the complex interactions of social class, social isolation, and unwillingness on the part of immigrants to use health care services. Fundamentally, epidemiologists and clinicians alike have not been prepared to take on the challenge of expanding suicide prevention beyond biomedical approaches. As with heart disease, if the basis of suicide is social and economic, the solution to suicide has to be social and economic.

A BRIEF HISTORY OF CVD PREVENTION

Writing about the Framingham Study in 1971, Gordon and Kannel said of CVD that "We are faced with a disease . . . which frequently attacks without warning, and in which the first symptoms are all too often the very last."[25(p1624)] They commented that even when heart disease becomes manifest clinically, individuals are rarely cured or return to full functionality. And, they urged, since heart disease can be asymptomatic even in its most severe form, prevention should become a priority even while technological advances were improving that increased an individual's chances of survival.

At the beginning of the Framingham Study, however, there was an unclear picture of the epidemiology of CVD. In fact, an epidemiological approach for a noninfectious disease was a novel concept in 1949, when the US Public Health Service decided to investigate possible predisposing conditions.[26] The prevailing wisdom of the time was that treating isolated systolic hypertension was dangerous, that elevated blood pressure in the elderly was normal, and that serum cholesterol was not a risk factor for CVD. Indeed, prior to President Roosevelt's massive stroke in 1945, his doctors recorded that his dangerously elevated blood pressure was "normal," a powerful example of the misconception of risk based solely on clinical observation.

As epidemiological studies began to provide more accurate appraisals of the natural history of heart disease, there were clinicians who began to suggest that, even in the absence of any clear demonstration of effectiveness, community-based programs that would modify lifestyles held the most promise for dramatically reducing morbidity and mortality due to CVD in persons aged younger than 65 years.[26] The term "risk factor" emerged from the Framingham Study to describe a modifiable attribute that predisposed individuals to CVD. As an increasing amount of data was marshaled that provided evidence of the interaction of multiple risk factors, there was an enormous shift in the way clinicians *and* the public health community addressed outcomes of complex, multifactorial etiologies. The importance of early identification of modifiable risk factors far distal to a deleterious outcome moved from the merely speculative stage to the recognition of the need for primary CVD prevention strategies to the development of interventions targeting population-level reductions of the precursors of heart disease.

As a result, it became unconscionable to consider as "prevention" interventions such as "clot busters" or coronary artery stents for individuals who appeared in emergency departments experiencing a myocardial infarction or acute chest pain. Moreover, there continued to be an unprecedented move away from a clinically oriented, high-risk approach to prevention, which entailed identifying the relatively small number of individuals who constituted the 2.5% of the population at highest risk. As such, they occupied the upper "abnormal" end of the normally dis-

tributed blood pressure curve, and intensive treatments could only hope to move them to the middle of the curve. Increasingly, clinicians were recognizing that cases of CVD do not arise among these high-risk subjects but rather from the "normal" blood pressure group[11] in accord with Rose's Theorem—that "a large number of people at small risk may give rise to more cases of disease than a small number who are at high risk."[12(p37)] Obviously, clinicians today continue to aggressively treat patients with hypertension or high cholesterol. Alone, the high-risk strategy identifies a minority of those individuals who die from cardiac disease and stroke, and it is palliative for those already identified as symptomatic, usually with temporary benefit. Only an alternative, radical approach aimed at shifting the entire population distribution of risk has accomplished significant reductions of CVD-related morbidity and mortality in some populations.[25]

SOCIAL MARKETING

In addition to fundamentally altering basic concepts in clinical care, during the early 1970s and into the 1980s there was an unprecedented number of large-scale efforts to change knowledge, attitudes, and health risk behaviors and to test interventions models for CVD prevention.[27–32] The first of these, the Stanford Three-Community Study and the North Karelia Project, began as media campaigns. Eventually, as evidence accumulated that changing knowledge, attitudes, and behaviors among individuals appeared to be most successful when cultural norms supported a healthy lifestyle, these became interventions to market environmental changes.[33] In addition to mass media, intensive community interventions targeted decreases in blood pressure, smoking, and composite risk for heart disease through free blood pressure screening and counseling and skills building and efficacy enhancement to promote behavioral change.

Prevention of CVD grew out of an atmosphere of public dread of heart disease and its related morbidities, such as the stroke that killed President Roosevelt in 1945. But while there was perceived social urgency, the really difficult environmental changes remained daunting compared with what was accomplished in these early demonstration projects.[34] Necessary structural changes that involve changing laws and policies that prohibit smoking at work and in public places, combating commercials that contain misleading nutritional information, and changing the public's attitudes toward exercise will require more than social marketing strategies. The field of CVD prevention, having moved from clinical treatment of risk factors to large-scale epidemiological studies to interventions that target social and cultural risk factors, now is faced with developing the most effective means of changing policy both in terms of clinical recommendations and widespread environmental changes.

LIMITATIONS OF CVD PREVENTION AS A MODEL

CVD as a "prevention role model" has its limitations as well. It is essential *not* to recapitulate the less successful elements of its history, especially the nearly exclusive early focus on middle-aged White men, with the apparent neglect of women and diverse ethnic groups, and a near lack of implementation of prevention strategies in less-developed countries.[35] For some sectors of mainstream America, dietary fat became an *accepted* public health enemy. But for many populations, such as African Americans,[36] Native Americans,[37] younger men,[38] and rural populations worldwide,[39,40] risk and protective factors remain understudied and interventions lag. In the United States and worldwide, subpopulations still exist that are vulnerable to CVD owing to lack of knowledge about, or cultural recognition and acceptance of, the risks and protective factors for CVD.

Most important, the effectiveness of community-based interventions for CVD has been called into question by studies carried out primarily in the 1980s, in which secular trends in the intervention communities reduced any apparent effects for some populations.[41–44] Despite this apparent lack of effectiveness, there are many who are convinced that were we to return to an emphasis on curative medicine applied to the individual, it would threaten to absorb public health into molecular medicine.[45] Pearson and Lewis[40] suggest that one of the reasons for failing to detect a large enough effect size in community intervention trials for CVD is that many of these trials have been carried out in "early adopter" communities (those communities that are first to implement novel interventions.) The intervention in most of these trials was a composite of education, screening, and risk assessment. However, from the work of Rogers and Shoemaker[46] on diffusion of interventions, we know that one of the characteristics of individuals who are early adopters is that they seek out new information.

In a similar fashion, education through mass media may be sufficient to alter the behavior of an early adopter community. Conversely, Pearson and Lewis suggest that in rural communities there has been a rise in CVD that may be due to characteristics of these communities that make them "late adopters" of CVD prevention.[40] Compared with the communities in which the major intervention trials for CVD have been carried out,[27–32] rural communities tend to comprise individuals who are underinsured or have no insurance and have a higher rate of poverty and a lower rate of educational attainment. These communities also have a different risk profile in terms of eating habits, traditionally consuming a high-fat, high-calorie diet that in the past was coupled with high caloric expenditure. With the advent of the mechanization of farming and other rural occupations and a more sedentary lifestyle, obesity is now a significant public health problem among inhabitants of rural communities.[40]

As a final consideration, the lack of clear-cut efficacy of community-based intervention trials for CVD probably relates to issues at theoretical, interventional, and evaluational stages.[47] Quite simply, the "preventive dose" may not have been large enough or the evaluation sensitive enough to detect shifts in the mean population risk toward lower levels.[48]

SUICIDE PREVENTION COMPARED WITH CVD PREVENTION

As Goldsmith and colleagues recently commented, "If ever a condition begged for an integrated understanding that takes into account biological, clinical, subjective, and social factors, this suicide prevention is it."[49(ix)] We feel strongly that in the United States, the sta-

tus of suicide prevention is analogous to preventive cardiology during the middle of the last century. Just as myocardial infarction was a "silent killer" then, present efforts toward suicide prevention in general remain focused on detecting or intervening just before or during the suicidal event (e.g., telephone crisis hotlines). Prevention of CVD underwent a transition from primarily clinically focused approaches to population-based approaches of prevention. The same has not been true of suicide prevention, which has never gone through a similar translational phase from clinical recognition of risk to population-based approaches based on prospective, longitudinal studies of risk factors. Many elements of the present dependence on interventions that focus on individuals in imminent danger of taking their own lives arose during debates of the 1930s and 1940s, primarily by psychoanalysts in the United States who saw each self-committed death as an individual or interpersonal act.[50] This perspective of suicide as an "individual act," however, gave shape to the course of suicide prevention during the 1970s (proliferation of suicide prevention centers,[51] whose volunteers knew little about the behavior and attitudes of the individuals seeking their services),[52] the 1980s (debates around the appropriateness of school-based early-recognition prevention programs, even while most youths and young adults who killed themselves were no longer in school),[53] and the 1990s (a near-absolute lack of outcome research), all of which stand in stark contrast to the course of preventive cardiology.

Some would argue that the translation from clinical knowledge of suicide risk to interventions for population-level risk reduction has in fact occurred. In a widely cited intervention implemented on the Swedish island of Gotland, primary care physicians were trained to recognize and treat depression.[54] Subsequently, reductions in depression-related morbidity were observed. Parenthetically, the researchers found a transient, statistically significant reduction in the suicide rate based solely on reductions in female suicides. Some have argued that this outcome was attributable to the intervention,[55] while others have viewed the result as a statistical fluctuation, negated if 5-year rolling mortality averages are used.[56] A well-designed replication with a larger sample that could be generalized to other populations does not exist, although the Gotland study is frequently cited as a model of suicide prevention.

Probably a better model of suicide prevention is the world's first nationally implemented, research-based suicide prevention implemented in Finland between 1992 and 1996.[4] However, because data were not collected that would have allowed for the control of confounding variables, it is difficult to conclude whether the program itself was responsible for the 20% reduction in suicide rates that occurred between 1991 and 1996 (note that the reduction began prior to the program's implementation).[57] Although school-based social skills training efforts to reduce suicidal behaviors are promising, they have not been tested in a rigorous fashion.[53] Longitudinal outcome data from community-level programs established in New Zealand and Canada for aboriginal populations are not yet available.[58,59] The only published prevention efforts shown to have a measurable impact on deaths have employed population-oriented approaches, such as the replacement of coal gas with less toxic North Sea gas in the United Kingdom[60] or recent changes in the packaging of paracetamol (acetaminophen) and salicylates, also in the United Kingdom.[61]

CREATING AN EPIDEMIOLOGY OF SUICIDE

In part, suicide prevention did not follow the same course as CVD prevention because of significant methodological challenges. Unlike the case with CVD, there is no similar, well-defined, prospectively developed epidemiology of suicide across the life course for any group. Key questions demand relevant data collected through prospective incidence studies of risk and protective factors for suicide. Investigators could (and should) enrich their samples with groups thought to bear key risk and protective factors. While appropriate precautions must be taken to protect those that may be at greater risk, the National Institute of Mental Health has released a report to address the ways in which practices standard to clinical trials can apply to suicidal patients as well.[62] Such data would permit us to test which risk or protective factors give rise to differential expression of morbidity, and perhaps mortality, captured in hypothesized "families" of related behaviors, events, and disorders. Without such groundwork, prevention efforts necessarily will "fly blind." Moreover, lack of a social-contextual model of suicide has deflated efforts to statistically predict who will kill themselves.

Many researchers may disagree with this appraisal. An array of retrospective studies, including our own,[63–87] as well as a few prospective studies,[88–93] have identified mental disorders and substance abuse as risk factors associated with suicide, with particular emphasis on depression, intoxication and chemical dependency, comorbid medical conditions, social isolation, unemployment and poverty, and stressful life events. It has been difficult (if not impossible) to incorporate measures of these conditions as accurate and useful screening or diagnostic "tests," in large part owing to their relatively low predictive value and the poverty of data available to assess the relative strengths of these risk factors in important but potentially distinctive subpopulations, such as men in their early adult years, women, adolescents, older adults, or members of diverse ethnic communities. Data are essential to provide the context for assessing the potential impact of protective factors that act in the presence of apparent risk factors to mitigate adverse outcomes. Many reported "risk profiles" for suicide really reflect uncontrolled findings from middle-aged White men.[94] Indeed, there are limited case–control studies of completed suicide that have been conducted for deaths among people aged 21 years and older on which to base risk assessment and prevention.[95–102]

Given this comparative context, we suggest that suicide prevention remains rooted in a traditional but limited approach, that of clinical treatment of risk factors, whether that is carried out at a population or individual level. The results thus far are limited in their generalizability. One prospective, naturalistic follow-up of 643 individuals treated with fluoxetine for depression found a nonsignificant reduction in risk of suicidal behavior.[103] There is some evidence that clozapine therapy reduces suicidal behaviors in patients with schizophrenia.[82] However, a study com-

paring clozapine therapy in patients with schizophrenia matched to a schizophrenic control group failed to find that clozapine treatment was associated with a reduced risk of completed suicide.[104] A controlled trial (the International Suicide Prevention Trial) to confirm these findings is currently under way.[104] Results from a study using retrospective data suggest that lithium treatment reduces suicide among bipolar patients,[105] but no prospective work has yet tested this important clinical observation. Zametkin et al.[106] have summarized the difficulties with predicting and preventing suicide in adolescents, particularly addressing the question of the efficacy of lithium and antidepressant pharmacotherapy for reducing suicide rates in this age group.

SUICIDE PREVENTION: ARE WE LATE ADOPTERS?

Taken together, how is the experience of CVD prevention best viewed to inform the evolution of suicide prevention? Suicide rates have been decreasing over the past decade in at least 1 population—US adolescents.[107] Could this be due to the fact that schools and other community organizations that serve adolescents have been (although unknowingly) "early adopters" of suicide prevention, while other communities have lagged behind? Perhaps it is due to a heightened degree of perceived social urgency, because of the tragic nature of a youthful suicide. Other communities may have less social capital to ensure the perception of social urgency following the death of one of their own. Just as Pearson and Lewis[40] have identified rurality as a risk factor for CVD, stigmatization of help-seeking behaviors for a mental health–related disorder or distress could represent a significant community risk factor for preventing suicide and related outcomes. Moreover, some marginalized communities, such as the seriously mental ill, the homeless, the unemployed, those involved with the courts and criminal justice system, and the elderly, have less access to mental health care and the means to pay for it.

Are stigmatization and marginalization the "risk" equivalents to poor education and low socioeconomic status in late adopter rural communities of CVD prevention? In both cases, rates of CVD and suicide have not demonstrated secular reductions and therefore represent important targets for population risk-reduction strategies, where effect sizes might be statistically significant, if the intervention efforts are rigorously evaluated. These interventions must be developed and implemented keeping in mind that education through mass media efforts will in all likelihood not be sufficient in late adopter communities. Additional barriers, such as lower awareness of symptoms in these populations, the lack of sufficient public health infrastructure to address these communities' needs, and the lack of political will to support funding for these marginalized groups, must all be considered. Using the terminology recommended by the Institute of Medicine[108] for preventive mental health, Table 1 provides examples of how different levels of interventions might be employed to implement programs for suicide prevention in some potentially late adopter communities.

THE US AIR FORCE AS A MODEL OF SUICIDE PREVENTION

One promising approach to suicide prevention is seen in the US Air Force.[109] In response to an alarming increase in suicide rates during the mid-1990s, top leadership man-

TABLE 1—The Language of Mental Health Prevention Applied to Preventing Suicide and Attempted Suicide

Intervention Terminology	Approach	Target	Objectives	Examples of Possible Future Prevention Efforts
Universal prevention strategies	Population	Implement sweeping, broadly directed initiatives in entire populations, not identified on the basis of individual risk. Develop programs that reach asymptomatic individuals.	Prevent disease through reducing risk and enhancing protective or mitigating factors.	1. Enhance school and community programs to reduce alcohol and substance abuse in youth and young adults. 2. Develop effective violence reduction programs among men aged 25–55 years. 3. Remove insurance barriers for access to mental health and substance abuse treatment.
Selective prevention strategies	High risk	Identify individuals or subgroups bearing a significantly higher than average risk of developing mental disorders or adverse outcomes.	Prevent disease through addressing population-specific characteristics that place individuals at higher than average risk.	1. Provide counseling and health services for homeless individuals and families. 2. Promote church-based and community programs to contact isolated elders. 3. Provide therapeutic support to victims of domestic violence.
Indicated prevention strategies	High risk	Identify high-risk individuals with detectable symptoms. Include asymptomatic individuals bearing defined risk markers.	Treat individuals with precursor signs and symptoms to prevent development of full-blown disorder.	1. Increase screening/treatment for depressed elders in primary care settings. 2. Treat elders with chronic pain syndromes more effectively. 3. Enhance lithium maintenance for persons with recurrent bipolar disorder. 4. Prescribe pharmacological therapies for individuals bearing biomarkers for psychiatric disorders associated with suicide and suicidal behaviors.

dated that suicide prevention had to become a communitywide Air Force responsibility, not solely a medical problem (Gen Thomas Moorman, oral communication, June 2001). A significant and sustained drop in suicide rates was observed following communitywide dissemination of the program.[109,110] Key components of the program were ongoing commitment from leadership, consistent and regular communication around the topic of suicide prevention, destigmatization of seeking help for a mental health problem, improved collaboration among community prevention agencies, and the identification and training of "everyday" gatekeepers.[111]

As a "model of cultural change," the Air Force prevention program potentially serves as the first demonstration of the relevance of Rose's Theorem for preventing suicide: improving overall community mental health can reduce the events of suicide more effectively than extensive efforts to identify the imminently suicidal individual.[110]

Although the Air Force community must be viewed as a select population, it may prove to be an excellent example of an early adopter community. As in CVD prevention, early adopter communities will have accelerated rates of population risk reduction for suicide and other outcomes. Nevertheless, experience with early adopter communities appears to be essential to inform the barriers (primarily in terms of social determinants) that must be overcome in order to successfully adapt interventions for late adopter communities worldwide.

CONCLUSION

By the early 1990s, investigators had begun to point out that, while there was a vast scholarly literature on the sociological, psychological, and biological aspects of suicide, prevention efforts had lagged considerably.[112–116] Given these observations and the current recognition of suicide as a global public health problem, what barriers still need to be addressed that will have an impact on the "prevention gap" that has come to characterize reducing suicide and suicidal behaviors?

Perhaps this gap derives from the fundamentally different perspectives of clinical and public health researchers. When clinicians do not observe manifest disease, they probably will conclude that treatment is not indicated. The epidemiologist, in contrast to the clinician, classifies individuals along a continuum of risk, favoring this approach to forcing a dichotomous distinction between "normal" and "abnormal." "Disease free" is not the same as "risk free." But the public health research community is not beyond critique. By dismissing suicide purely on the basis of a low incidence rate, the opportunity to affect the wider array of related conditions concerned with destructive behaviors has been largely ignored. Highly associated morbidities for CVD were not recognized half a century ago, either.

Clinicians, ignoring the public health nature of suicide, rely primarily on their ability (albeit limited) to change an individual's suicidal behavior. In this sense they have promoted, however unwittingly, the social isolation of the community problems of suicide and suicidal behaviors. In addition, psychopathologists have failed thus far to discern those factors that protect most people with severe psychiatric disorders from attempting or completing suicide.[115] Clinicians tend not to see at-risk individuals whose protective factors have effectively insulated them from manifesting signs or symptoms of illness. Inevitably, exploring the nature of protective factors requires engaging the public health community, an opportunity not yet exploited. Too often, clinicians and public health professionals have held fast to their respective worldviews. How can we identify ways to move the field forward through a synthesis of the 2 approaches?

Like recent collaborative prevention efforts from other fields,[116–120] the greatest CVD prevention successes still appear to have been community driven. Suicide prevention efforts that would target communities that bear higher than usual risk for suicide must encompass older adults, the homeless, adolescents in turmoil, prisoners, or the severely and persistently mentally ill. Undoubtedly, these will require novel approaches to engage their members, as many of these individuals do not readily contact public mental health systems or practicing clinicians. As an example, Project Link is a university-led consortium of 5 community agencies in Monroe County, New York.[121] The program is distinctive in its nontraditional delivery of mental health services to severely mentally ill adults involved with the criminal justice system. The emphasis is on providing services to individuals in courtroom and jail settings. Preliminary outcome data suggest that Project Link may be effective in reducing recidivism and in improving community adjustment among severely mentally ill patients with histories of arrest and incarceration.[121] Community is not just the sum of its citizens—rather, it is the web of relationships between people and institutions.[122] Other "communities" that hold promise as intervention sites include large corporations, police and fire departments, diverse ethnic communities, governmental agencies, universities, and military services worldwide.

Efforts to prevent cardiovascular disease helped set the standard for conducting community-based interventions. We would argue that the history of CVD prevention is best considered within the context that interventions implemented in a community environment must always address the considerable "noise" of real-life circumstances.[123] Clinicians and epidemiologists in the United States are faced with the challenges inherent to the study of behavioral change in population laboratories. Imperfect methodologies have resulted in mixed results on the effectiveness of interventions for CVD prevention. Psychiatric research now has begun moving toward a more inclusive, community-based approach. But implementing preventive mental health strategies in real-world community settings could greatly benefit from understanding the successes and failures of prevention strategies developed for other outcomes whose origins are largely societal. Developing population risk reduction approaches for suicide, through prevention of its precursors in communities, could result in truly innovative (and potentially effective) programs for suicide prevention. ■

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K.L. Knox led the writing of the article and reviewed the literature. Y. Conwell contributed substantially to revision of the article. E.D. Caine conceived of using the prevention of heart disease as a framework for assessing the current status of suicide prevention. All authors contributed to revisions of the article.

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